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## Spontaneous splenic rupture: a rare complication of Q fever in Australia

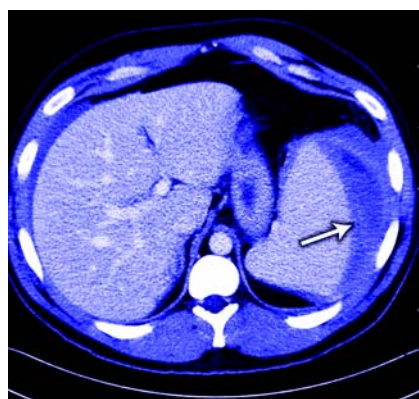
Amanda J Wade, Tim Walker, Eugene Athan and Andrew J Hughes

**TO THE EDITOR:** Q fever is a serious disease caused by *Coxiella burnetii*, and usually occurs in people exposed to livestock. In Australia, acute Q fever generally manifests as nonspecific febrile illness. We report a case of spontaneous splenic rupture as a complication of acute Q fever acquired in Australia.

A 29-year-old, previously well man presented in March 2005, with 5 days of fever, rigors and severe headache. On examination he had a temperature of 40°C, was tachycardic at 100 beats per minute, but normotensive. There were no focal examination findings. Investigations revealed thrombocytopenia at  $123 \times 10^9/L$ , a normal white cell count, and clear chest x-ray. The patient worked at a factory that processed animal placentas and fetal products. A provisional diagnosis of Q fever was made based on the illness and this exposure. The man was admitted and treated with empiric doxycycline, penicillin, and ceftriaxone.

On Day 1 of the admission, he developed sudden, severe, left upper quadrant abdominal pain with shoulder tip radiation, diaphoresis and hypotension. An urgent computed tomography scan of the abdomen revealed splenomegaly with a diameter of 14 cm, and a crescentic, subcapsular splenic haematoma with rupture

### Abdominal computed tomography scan



A crescentic, subcapsular splenic haematoma is visible (arrow). ◆

into the peritoneal space (Box). There was no history of trauma. He was admitted to the high dependency unit for monitoring. His haemoglobin level dropped from 151 g/L to 102 g/L, but he was managed conservatively and discharged from the high dependency unit 24 hours later.

Acute Q fever was confirmed by polymerase chain reaction on Day 2, and antibiotic therapy was simplified to doxycycline 100 mg twice daily for 14 days. He was discharged from hospital on Day 5, and recovered fully. Seroconversion to Q fever was subsequently confirmed. His workplace now practices Q fever prevention policies, including pre-employment vaccination.

Common presentations of Q fever include nonspecific febrile illness, pneumonia and

hepatitis. There are five reported cases of Q fever associated with spontaneous splenic rupture, but this is the first Australian case.<sup>1-5</sup> The other patients presented with flu-like symptoms and abdominal pain of 2–14 days' duration, and required splenectomy between Day 1 and 4 of admission.

Clinicians should be aware of splenic rupture as a potential complication of Q fever in Australia.

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## Prothrombinex use for the reversal of warfarin: is fresh frozen plasma needed?

Julie H Crawford and  
Bradley M Augustson

**TO THE EDITOR:** Current Australian guidelines for urgent warfarin reversal recommend withholding warfarin, and giving vitamin K as well as factor replacement (Prothrombinex HT; PTX) with or without fresh frozen plasma (FFP).<sup>1</sup> PTX administration without FFP is recommended only when FFP is unavailable, as PTX factor VII levels are low and unquantified.<sup>2</sup> Transfusion of 25–50 IU/kg PTX and 150–300 mL of FFP is advised. We examined the effectiveness of warfarin reversal using PTX alone or combined with FFP.

One hundred and fifty-five patients given PTX were identified from the transfusion medicine unit's computer records. Fifty patients were excluded — no warfarin (19), non-hospital patients (11), no record of PTX administration (14), no monitoring (5), normal international normalised ratio (INR) (1).

One hundred and five patients who had received warfarin were reviewed. The reasons for anticoagulation were atrial fibrillation (45 patients), venous thromboembolism (22 patients), cardiac valve replacement (21 patients), myocardial

### 1 International normalised ratio (INR) before transfusion of Prothrombinex HT (PTX) and dose of PTX in patients in the two groups (PTX and PTX + fresh frozen plasma [FFP]) at increasing levels of anticoagulation

INR before PTX	PTX group (n = 74)			PTX + FFP group (n = 31)			
	No. of patients	PTX dose IU (range)	PTX IU/kg* (range)	No. of patients	PTX dose IU (range)	PTX IU/kg† (range)	FFP units (range)
<2.0	8	556 (500–1000)	7.69 (5.32–11.1)	1	500	9.09	2
2.0–3.9	22	929 (500–2000)	12.81 (4.85–31.25)	16	562 (500–1000)	8.48 (5.1–17.54)	2.2 (1–10)
4.0–5.9	12	1083 (500–3000)	16.2 (6.67–34.8)	7	1000 (500–2000)	15.51 (7.14–44.4)	4 (2–6)
6.0 +	32	1187 (500–2000)	14.17 (4.17–27.03)	7	929 (500–1000)	12.65 (7.69–19.23)	4.7 (2–10)

\* PTX doses calculated for the 89% of patients in the PTX group whose weight was recorded in notes.

† Doses calculated for the 77% of patients in the PTX + FFP group whose weight was recorded in notes. ◆

infarction (9 patients), and other (8 patients). PTX was administered for bleeding in 51 patients (32 had major bleeds), suspected bleeding in eight patients, high INR in 11 patients, and before a procedure in 35 patients. Bleeding severity was graded using published criteria.<sup>3</sup> Post-treatment INR and patient details were used to determine clinically significant INR lowering and haemostasis.

The patients were divided into two groups — patients administered PTX without FFP (n = 74) and patients administered PTX and FFP (n = 31) (Box 1). Seventy-four patients (71% receiving PTX; 67.7% PTX + FFP) were given vitamin K (71 intravenously) to ensure ongoing correction of coagulopathy. This was administered within 2 hours of PTX (77%), with doses 1–2.5 mg (44 patients),

### 2 Absolute and percentage reduction in international normalised ratio (INR) per vial of Prothrombinex HT (PTX) transfused for the 74 patients in the PTX group (treated with PTX without fresh frozen plasma)

INR before PTX		INR after PTX					
		< 1.5	1.5–1.9	2.0–2.9	3.0–3.9	4.0–5.9	6.0 +
<2.0	No. of patients (no. with bleeding)	5 (0)	3 (1)	—	—	—	—
	Mean percentage reduction in INR (range)	18.6% (7.1%–31.3%)	9.3% (0–16.7%)	—	—	—	—
2.0–3.9	No. of patients (no. with bleeding)	7 (4)	9 (2)	6 (1)	—	—	—
	Mean percentage reduction in INR (range)	35.9% (21.4%–53.6%)	23.5% (9.1%–44.4%)	30.5% (9.6%–40.0%)	—	—	—
4.0–5.9	No. of patients (no. with bleeding)	1 (1)	7 (5)	3 (3)	1 (1)	—	—
	Mean percentage reduction in INR (range)	33.8%	34.4% (9.9%–65.5%)	31.7% (25.5%–39.5%)	21.8%	—	—
6.0 +	No. of patients (no. with bleeding)	8 (4)	4 (1)	8 (5)	8 (2)	2 (1)	2 (0)
	Mean percentage reduction in INR (range)	52.8% (19.4%–86.4%)	66.9% (44.5%–90.1%)	55.7% (33.6%–82.5%)	37.8% (19.9%–40.2%)	39.6% (35.7%–43.5%)	26.7% (19.0%–34.4%)

## LETTERS

3–6 mg (13 patients), and 10 mg (17 patients).

The mean PTX dose used was 862 IU or 12.9 IU/kg. Slightly higher PTX doses were administered to patients in the PTX group (913 IU or 13.1 IU/kg) than to patients receiving PTX and FFP (742 IU or 12.3 IU/kg) (Box 1). Seven of 89 patients (7.9%) received the published recommended PTX dose.<sup>1</sup> The remaining patients received < 25 IU/kg. Neither the degree of warfarin coagulopathy nor FFP transfusion appeared to influence PTX doses administered. One patient in the PTX group with end-stage renal impairment failed to show any reduction in INR, but did achieve haemostasis. All patients with bleeding achieved haemostasis after PTX.

The degree of INR correction after PTX varied considerably, with the percentage reduction in INR after PTX ranging from 0 to >90% per vial of PTX transfused (Box 2). Achievement of haemostasis did not require normalisation of INR (Box 2). The INR is

designed for monitoring therapeutic warfarin levels and its accuracy declines with excessively prolonged clotting times. As PTX is more likely to produce correction of coagulopathy through increased thrombin rather than through factor VII replacement, INR may not be the best test for monitoring haemostatic changes after PTX.

Our data suggest that FFP may be unnecessary when PTX is used to reverse warfarin coagulopathy. We have also shown that doses of PTX under 25 IU/kg are effective. Administration of low-dose PTX will produce cost savings and may reduce PTX-associated adverse events (including infusional reactions, thrombocytopenia [secondary to heparin], and thrombosis). The risks associated with FFP transfusion (including allergic reaction, transfusion-related acute lung injury, fluid overload, and infection transmission) would be eliminated. A prospective study is necessary to confirm our findings and assess the efficacy of lower PTX

doses, so that, if our results are confirmed, warfarin reversal guidelines can be reviewed.

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## Reversible obesity-related glomerulopathy following weight reduction

Huy A Tran

**TO THE EDITOR:** Obesity-related glomerulopathy (ORG) is a condition that is partially reversible by weight loss.

A 48-year-old morbidly obese man presented with massive proteinuria (8.4 g/day; reference range[RR], <0.03 g/day). His medical history included hypertension of 4 years, morbid obesity, gout and impaired fasting glycaemia. His hypertension had been relatively well controlled, with no known end-organ complication until the current review. His urinary protein excretion rate measured 6 months earlier had been 0.13 g/day. Long-standing medications included aspirin, lisinopril and allopurinol.

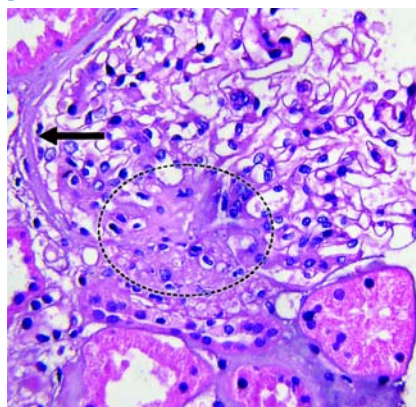
The patient weighed 125 kg and was 1.77 m in height (body mass index, 40 kg/m<sup>2</sup> [class III obesity]). His blood pressure was 130/70 mmHg, with no postural hypotension. The fundi were normal, with no evidence of hypertensive retinopathy. There was no peripheral pitting oedema and there were no tendon xanthomata.

A repeat urinary protein measurement showed an excretion rate of 7.9 g/day, confirming the earlier result. Renal and liver function tests were normal. Other laboratory tests produced the following results: fasting cholesterol, 5.5 mmol/L (RR, <4.0 mmol/L); triglycerides, 2.4 mmol/L (RR, <1.8 mmol/L); serum creatinine, 92 µmol/L (RR, 60–110 µmol/L); albumin, 38 g/L (RR, 35–45 g/L); fasting glucose, 6.8 mmol/L (RR, <5.5 mmol/L); serum insulin, 66 mU/L (RR, 5–25 mU/L); and C-peptide, 6.8 nmol/L (RR, 0.2–0.6 nmol/L). All relevant tests for glomerulonephritis, including serum antinuclear antibodies, antinuclear cytoplasmic antibodies, C3 and C4 levels, protein electrophoretic studies, and hepatitis B and C serology were negative, making a diagnosis of glomerulonephritis unlikely.

While awaiting the renal biopsy, the patient decided to attempt rapid weight loss. He began consuming mostly one meal a day of about 5000 kJ (which included about 2000 kJ of protein). Six weeks later, his weight had fallen to 118 kg and his blood pressure was 130/80, with no oedema. His urinary protein excretion rate was 7.6 g/day.

After 18 weeks, the patient weighed 110 kg and his urinary protein excretion rate had fallen to 2.1 g/day. At 6 months, his weight loss had plateaued at 102 kg and urinary protein excretion was 0.85 g/day.

### Histopathology of a representative glomerulus



*There is prominent vascular pole/perihilar sclerosis (broken circular line) and mild fibrosis of the Bowman capsule (arrow) (haematoxylin–eosin stain; original magnification, × 40). No evidence of hypertensive diabetic nephropathy or tubular disease was detected. Other histological findings (not shown here) included glomerulomegaly, peripheral hyalinosis and mild basement membrane thickening.<sup>1</sup> Morphologically, obesity-related glomerulopathy can be difficult to separate from idiopathic focal and segmental glomerulosclerosis.<sup>1</sup>* ◆

The renal biopsy showed features consistent with ORG<sup>1</sup> (Box). No other abnormality was detected. A concomitant minimal change lesion with spontaneous remission would be a possible explanation, but such a lesion is exceedingly rare and difficult to exclude in the absence of electron microscopy. It is also unlikely given the patient's age and abnormal light microscopy finding.

It is apparent that class III obesity, through unknown mechanisms,<sup>2</sup> is a major contributing factor in causing ORG and massive proteinuria — conditions that can be readily improved by weight loss. It remains to be seen in this case whether further weight reduction will further reduce the proteinuria, which, on the other hand, may also be confounded by the hypertension. Impaired fasting glycaemia is not a cause of proteinuria.<sup>3</sup>

This clinical vignette reinforces the need to check vigilantly for proteinuria and rigorously advise obese patients to attempt weight reduction, especially now that obesity has been confirmed to be an independent risk factor for end-stage renal disease.<sup>4</sup>

### Acknowledgement

I would like to sincerely thank Dr P Woodford for his assistance with the provision and histological interpretation of the renal biopsy.

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## Establishment of an Australian motor neurone disease registry

Matthew C Kiernan, Paul Talman, Robert D Henderson and Rodney Harris, on behalf of the AMNDR Steering Committee

**TO THE EDITOR:** Motor neurone disease (MND) is a relentlessly progressive neurodegenerative disease with a median survival of 1–3 years. It results in the death of nearly 400 Australians per year.<sup>1</sup> The management of MND remains problematic, tending to be offered in a heterogeneous and ad-hoc fashion across Australia. This heterogeneity arises in part from a lack of understanding of the aetiology of the disease and its progression in different patients,<sup>2</sup> the absence of established guidelines for standard care,<sup>3</sup> and a lack of concentrated experience among medical practitioners, nursing and allied health care workers in treating patients with MND.

With new diagnostic techniques, treatments and interventions for MND undergoing trials, there is a clear need for more baseline information on MND management and outcomes and a method for monitoring any changes on a population basis.<sup>4</sup> To facilitate this, the Australian Motor Neurone Disease Registry (AMNDR) has been developed. The Registry is governed by a steering committee comprising specialist physicians from each state and territory around Australia, neuroscientists, an epidemiologist and patient representatives.

AMNDR was launched in Sydney in June 2004 to coincide with Motor Neurone Dis-

## Demographic data for 351 patients with motor neurone disease enrolled in the Australian Motor Neurone Disease Registry to November 2005, by phenotype

Phenotype, site of symptom onset	Percentage of all registrations*	Mean age at onset in years (SD)	Sex ratio (F : M)	Mean survival time in months (SD; range) <sup>†</sup>	Deaths (%)
<b>Global<sup>†</sup></b>					
Bulbar	27%	64 (13)	1.5 : 1	27 (10; 20–48)	24%
Cervical	25%	55 (19)	0.4 : 1	20 (10; 7–33)	13%
Lumbar	19%	61 (10)	1.1 : 1	36 (16; 14–63)	24%
<b>Flail limb<sup>§</sup></b>					
Arm	10%	63 (13)	0.07 : 1	95 (85; 30–238)	27%
Leg	10%	58 (10)	2 : 1	59 (28; 33–100)	7%
<b>Primary lateral sclerosis<sup>¶</sup></b>					
All regions	11%	54 (9)	0.9 : 1	103 (55; 24–259)	0

\* Sum > 100% because of rounding. † Censored at November 2005.

‡ Global phenotype = combined upper and lower motor neurone signs in at least two spinal regions.

§ Flail limb variants = predominantly lower motor involvement of arms or legs, with prolonged disease duration.

¶ Primary lateral sclerosis = characterised by pure upper motor neurone involvement.

ease Global Awareness Day and, to November 2005, had enrolled 351 patients from 67 study locations, with 88% of registrations coming from 10 major sites. As of September 2005, 90 patients had undergone at least one further follow-up assessment. A copy of the registration, assessment and completion case report forms can be viewed on the AMNDR website (<http://www.amndr.org.au>).

From the patient information collected in the registration data, three distinct clinical phenotypes have emerged (Box). Patients with the “global” MND phenotype (combined upper and lower motor neurone signs in at least two regions) tended to have the shortest survival time.

AMNDR was designed to obtain information that will increase the understanding of MND and its progression in different patients. Through the process of registry establishment, those involved in care, management and scientific research related to MND have been galvanised into a more effective and cooperative working unit. Ownership of the database has been retained by all who have contributed to data collection, and the information has been increasingly used to promote research into the causes and treatment of MND, locally and internationally.<sup>5</sup> Through participation, it is expected that treating doctors will be able to evaluate their current management and associated patient outcomes relative to other centres around Australia.

### Acknowledgements

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